Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Discovery of Urinary Branched Chain Amino Acids as a Marker of Proximal Tubular Metabolic Dysfunction in Acute Kidney Injury: A Metabolomic and Transcriptomic Integration

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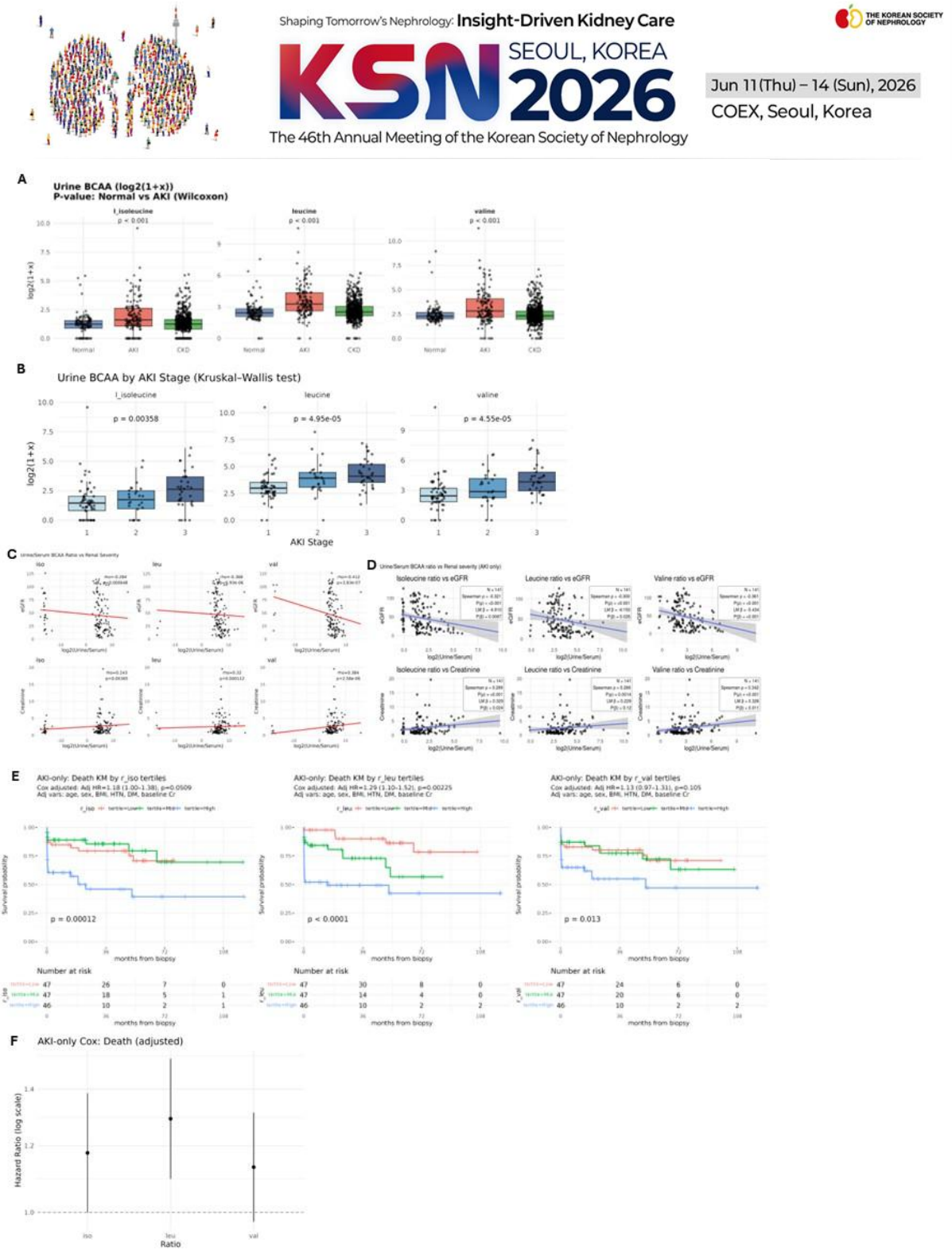
Objectives : Acute kidney injury (AKI) induces profound metabolic reprogramming within the proximal tubule. We aimed to identify key metabolic signatures of kidney injury and clinical outcomes through a discovery-driven approach using a prospective multicenter cohort.
Methods : This study utilized data and samples from KORNERSTONE, a prospective multicenter kidney disease biobank in Korea. Untargeted plasma and urinary metabolomic profiling identified metabolites differentially expressed across KDIGO stages. After identifying BCAA dysregulation as the most prominent metabolic shift, we performed single-cell transcriptomic analysis using datasets GSE252111 and GSE202109 to examine proximal tubule-specific molecular mechanisms.
Results : Untargeted screening identified urinary BCAAs (isoleucine, leucine, and valine) as the top-ranking metabolic signatures, exhibiting significant elevation in AKI patients ($P < 0.001$) with a clear dose-response relationship across KDIGO stages. Crucially, while urinary BCAA levels were highly sensitive to kidney injury, plasma BCAA profiles showed no significant correlation with kidney function or injury severity. This divergence indicates that urinary BCAA elevation is not a reflection of systemic metabolic changes but rather a specific "leak" resulting from local tubular failure. Single-cell transcriptomic characterization confirmed this mechanism, revealing a marked and coordinated suppression of proximal tubule-specific BCAA transporters (e.g., SLC7A8, SLC7A9) and catabolic enzymes in injured cells. GSEA further demonstrated a global collapse of high-efficiency energy pathways, including oxidative phosphorylation and fatty acid metabolism, in injured proximal tubules. Higher urinary BCAA levels were independently associated with an increased risk of mortality ($P = 0.00012$ for isoleucine tertiles), demonstrating their strong prognostic value.
Conclusions : Through untargeted metabolomic discovery, we identified urinary BCAAs as a specific and sensitive indicator of proximal tubular metabolic and transport failure in AKI. These findings, independent of systemic plasma levels, suggest that urinary BCAAs serve as a clinically relevant window into kidney metabolic reprogramming and adverse patient outcomes within the KORNERSTONE prospective cohort.

slide1.JPG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A
Urine BCAA (log2(1+x))
P-value: Normal vs AKI (Wilcoxon)
B
Urine BCAA by AKI Stage (Kruskal–Wallis test)
AKI Stage
C
D
E
AKI-only: Death KM by r_iso tertiles
AKI-only: Death KM by r_leu tertiles
AKI-only: Death KM by r_val tertiles
months from biopsy
Number at risk
F
AKI-only Cox: Death (adjusted)
Hazard Ratio (log scale)
Ratio

slide1.JPG

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